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Uric acid-lowering activity and mechanisms of Chinese medicines with medicine-food homology: a systematic study

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ARTICLE INFO

Article history
Received 05 September 2024
Accepted 26 November 2024
Available online 25 December 2024

Keywords
Hyperuricemia (HUA)
Medicine food homology
Chinese medicine
Active compounds
Extract

ABSTRACT

Objective To summarize the uric acid-lowering effects and mechanisms of Chinese medicines with medicine-food homology, aiming to provide novel perspectives for the development of new anti-hyperuricemia (HUA) drugs.

Methods Papers on the research of HUA prevention and treatment with medicine-food homology from December 15, 2002 to August 10, 2024 were screened and collected through China National Knowledge Infrastructure (CNKI), PubMed, ScienceDirect, and Google Scholar. Subsequently, the impact of these medications and their extracts, as well as the active compounds on HUA were assessed.

Results A total of 148 relevant papers were collected, including 43 kinds of Chinese medicines and 61 active compounds, all of which have anti-HUA activity. Among them, 41 kinds of Chinese medicines could inhibit the activity of xanthine oxidase, thus leading to the inhibition of uric acid production; and 22 kinds of Chinese medicines could facilitate uric acid excretion, while 15 kinds of Chinese medicines could reduce the inflammation levels in the body and promoting renal protection. Notably, polyphenols and flavonoids are the key active components for the uric acid-lowering effects.

Conclusion This study systematically summarized and analyzed the uric acid-lowering effects and mechanisms of Chinese medicines with medicine-food homology, laying a foundation for their development as HUA agents.

1 Introduction

Hyperuricemia (HUA) is a metabolic disorder caused by excessive production of uric acid (UA), insufficient excretion of UA, or a combination of both [1]. The final product of purine adenosine metabolism in a body is UA, and xanthine oxidase (XOD) is the pivotal enzyme in the formation of UA during this process (Figure 1). Upon synthesis, UA is transported to the kidneys for excretion via transporters like adenosine triphosphate (ATP)-binding cassette subfamily G member 2 (ABCG2), urate transporter 1 (URAT1), and glucose transporter 9 (GLUT9). When the activities of XOD, URAT1, and GLUT9 increase, while the activity of ABCG2 decreases, it will lead to a reduction in UA excretion and an elevation in serum UA levels, which can easily trigger HUA. It is widely

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Peer review under the responsibility of Hunan University of Chinese Medicine.

DOI: 10.1016/j.dcmed.2025.01.004

Citation: QIN FY, ZHU YS, ZHAO LW, et al. Uric acid-lowering activity and mechanisms of Chinese medicines with medicine-food homology: a systematic study. Digital Chinese Medicine, 2024, 7(4): 405-418.

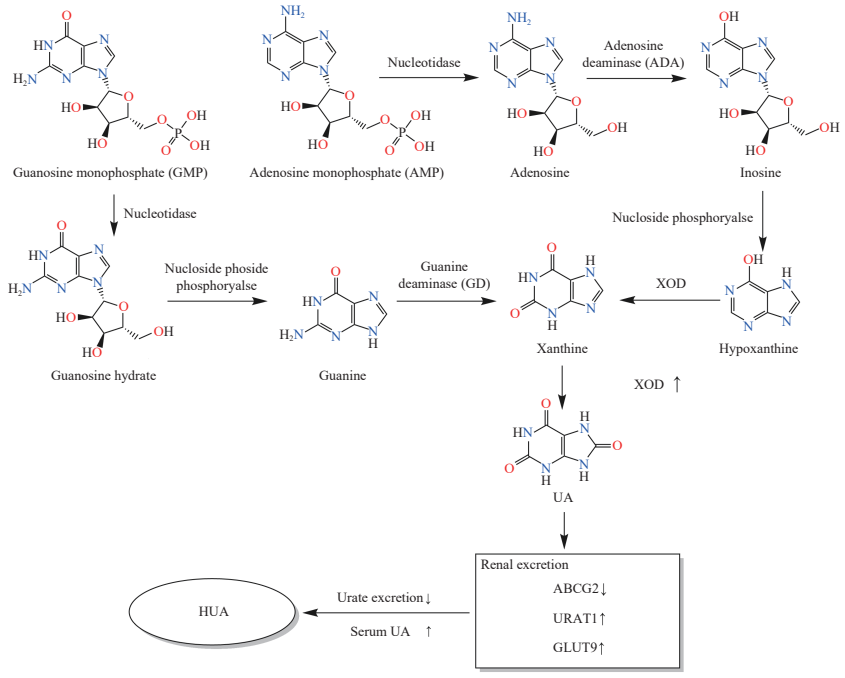

Figure 1 Schematic representation of HUA development

acknowledged that if the blood UA levels are higher than the normal threshold (male > 420 μmol/L, female > 360 μmol/L), it should be classified as HUA [2]. However, with the progress of social development and healthcare improvement, HUA has become a health issue worldwide.

With the improvement of living standards, diets tend to be high in sugar and purine, showing an increasing trend in HUA cases. Epidemiological studies have indicated that the prevalence of HUA in China alone has reached 13.3% [3], with an annual increase. Moreover, the condition is becoming more prevalent among younger populations [4], ranking it as the fourth most common metabolic disorder following hypertension, hyperlipidemia, and hyperglycemia [5]. Early stage HUA often shows no symptoms, but in advanced stages it can cause metabolic complications, which pose a substantial threat to public health and makes timely prevention and treatment of utmost importance. The increasing incidence of HUA highlights the need for the development of medications with low toxicity and high efficacy property.

The treatment of HUA in modern medical practice mainly focuses on three aspects (Figure 2): inhibiting XOD activity to reduce UA production with medications (such as allopurinol); blocking UA transporters to increase UA excretion with drugs (such as benzbromarone); facilitating the conversion of UA to decrease its accumulation with common agents (such as rasburicase). However, in current clinical practice, numerous western medications used for treating HUA exhibit considerable toxicity and side effects. Allopurinol, an XOD inhibitor, is linked to adverse drug reactions (ADRs) that may result in dermatological, gastrointestinal, and systemic complications [6, 7]. Severe cutaneous adverse reactions (SCARs) [6], Stevens-Johnson syndrome (SJS), and toxic epidermal necrolysis (TEN) [8] with a mortality rate of 20% – 28%. Benzbromarone, a drug that enhance UA excretion, shows mitochondrial toxicity in hepatocytes and contributes to liver damage through its metabolites and genetic polymorphisms [9]. Rasburicase, which promotes UA degradation, often triggers allergic responses, hemolysis, and methemoglobinemia due to its immunogenicity [10].

To find effective, low-toxicity, and cost-efficient treatments for HUA, researchers are focusing on natural products. Bioactive compounds extracted from medicinal plants can influence UA metabolism and enhance the body's barrier function through the inhibition of key enzymes involved in UA synthesis, regulation of UA transporter protein expression, reduction of systemic inflammatory responses, and modulation of gut microbiota. These compounds possess significant potential for the prevention and treatment of HUA. Additionally, there is a close correlation between HUA and diet, and modifying the diet may contribute to the alleviation of its symptoms [11]. Chinese medicines with medicine-food homology

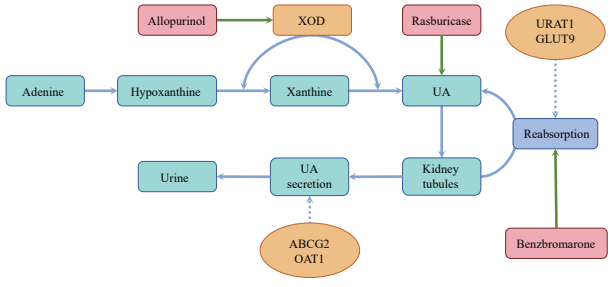

Figure 2 Three main treatment modalities for HUA and their representative medications

OAT1, organic anion transporter1.

offer promising potential in the treatment of HUA and serve as therapeutic agents for regulating physiology and preventing disease. Therefore, this review aims to systematically summarize the research progress on Chinese medicines with medicine-food homology in treating HUA, focusing on their active compounds, mechanisms of action, and therapeutic potential.

2 Methods

In this study, a systematic review and synthesis of relevant reports on the research into the prevention and treatment of HUA with medicine-food homology from December 15, 2002 to August 10, 2024 were screened and collected, through China National Knowledge Infrastructure (CNKI), PubMed, ScienceDirect, and Google Scholar. Retrieval keywords, such as "HUA" "UA lowering" "gout" "medicine-food homology" and "extract" were included. Medications that might potentially induce HUA, as well as primary active drugs and literature related to Chinese herbal compound prescriptions, multi-drug combinations, and Chinese herbal compound beverages with unclear compositions were excluded in this study. Subsequently, the impact of these medication, and their extracts, as well as the active compounds on HUA were assessed.

3 Results

A total of 148 relevant papers were collected, including 43 kinds of Chinese medicines (Table 1) and 61 active compounds (Figure 3), all of which have significant efficacy or potential in treating HUA. The primary mechanisms of action for these herbal medicines can be classified into three key categories: inhibition of UA production, enhancement of UA excretion, and reduction of systemic inflammation levels to protect renal functions. Specifically, 41 kinds of Chinese medicines exhibited inhibitory effects on XOD activity, effectively suppressing UA synthesis. Additionally, 22 kinds of Chinese medicines promoted the excretion of UA, while 15 kinds of Chinese medicines displayed anti-inflammatory properties, which contribute to the preservation of kidney health.

3.1 Chinese medicine for inhibiting UA production

XOD is crucial for UA production, and the inhibition of XOD can significantly reduce UA synthesis. Therefore, XOD inhibitors are a key focus in developing therapeutic strategies for HUA. Studies showed that several herbs, including Bohe (Sophorae Flos) [12], Huaihua (Sophorae Flos) [12-16], Juemingzi (Cassiae Semen) [17, 18], Machixian (Portulacae Herba) [19], Zhijuzi (Semen Hoveniae) [20-24], Baihe (Lilii Bulbus) [25], Dingxiang (Caryophylli Flos) [26], Shanyao (Dioscoreae Rhizoma) [27], Xiaohuixiang (Foeniculi Fructus) [28], Huoxiang (Pogostemonis Herba) [29], Shanzha (Crataegi Fructus) [30-32], Baizhi (Angeljcae Dahuricae Radix) [33], Xiakucao (Prunellae Spica) [34], Tiepishihu (Dendroii Officinalis Caulis) [35], Xihonghua (Croci Stigma) [36-38], Fuling (Poria) [14, 39], Huangqi (Astragali Radix) [25], Duzhongye (Eucommiae Folium) [48], Gouqizi (Lycii Fructus) [49], Gaoliangjiang (Alpiniae Officinarum Rhizoma) [13, 52, 53], Ganjiang (Zingiberis Rhizoma), Shengjiang (Zingiberis Rhizoma Recens) [27, 54, 55], Roudoukou (Myristicae Semen) [56, 57], Gegen (Puerariae Lobatae Radix) [30, 58], and Juhua (Chrysanthemi Flos) [30, 60], exhibit significant XOD inhibitory activities. Pugongying (Taraxaci Herba) [44, 66-68] and Yuganzi (Phyllanthi

Table 1 Mechanisms of action of Chinese medicine on HUA

Chinese medicine	Mechanism	Reference
Bohe (Menthae Haplocalycis Herba)	XOD↓	[12]
Huaihua (Sophorae Flos)	XOD↓	[12-16]
Juemingzi (Cassiae Semen)	XOD↓, UA↓	[17, 18]
Machixian (Portulacae Herba)	XOD↓, UA↓	[19]
Zhijuzi (Semen Hoveniae)	XOD↓	[20-24]
Baihe (Lilii Bulbus)	XOD↓	[25]
Dingxiang (Caryophylli Flos)	XOD↓, UA↓	[26]
Shanyao (Dioscoreae Rhizoma)	XOD↓, UA↓	[27]
Xiaohuixiang (Foeniculi Fructus)	XOD↓	[28]
Huoxiang (Pogostemonis Herba)	XOD↓	[29]
Shanzha (Crataegi Fructus)	XOD↓, UA↓	[30-32]
Baizhi (Angeljcae Dahuricae Radix)	XOD↓	[33]
Xiakucao (Prunellae Spica)	XOD↓, UA↓	[34]
Tiepishihu (Dendroii Officinalis Caulis)	XOD↓, URAT1↓, ABCG2↓, GLUT9↓	[35]
Xihonghua (Croci Stigma)	XOD↓, UARTI↓, GLUT9↓, ABCG2↓	[36-38]
Fuling (Poria)	XOD↓, UA↓, OAT1↑, OAT2↑, OAT3↑, UARTI↓, GLUT9↓	[14, 39-43]

Table 1 Continued

Chinese medicine	Mechanism	Reference
Huangqi (Astragali Radix)	XOD↓, UA↓, Cr↓, BUN↓, ALT↓, AST↓	[25, 44-47]
Duzhongye (Eucommiae Folium)	XOD↓, UA↓, Cr↓, BUN↓, IL-6↓, TNF-α↓, TLR4↓, GLUT9↓	[48-50]
Gouqizi (Lycii Fructus)	XOD↓, UA↓, IL-1β↓, TNF-α↓, GLUT9↓, OAT1↑, OAT3↑	[49, 51]
Gaoliangjiang (Alpiniae Officinarum Rhizoma)	XOD↓, URAT1↓, GLUT9↓, Cr↓, BUN↓, MDA↓	[13, 52, 53]
Ganjiang (Zingiberis Rhizoma)/Shengjiang (Zingiberi Rhizoma Recens)	XOD↓, UA↓, IL-1β↓, IL-6↓, TNF-α↓, COX-2↓	[27, 54, 55]
Roudoukou (Myristicae Semen)	XOD↓, UA↓, TNF-α↓	[56, 57]
Gegen (Puerariae Lobatae Radix)	XOD↓, ALP↑, ABCG2↑	[30, 58, 59]
Juhua (Chrysanthemi Flos)	XOD↓, XDH↓, UA↓, IL-1β↓, GLUT9↓, URAT1↓, OAT1↑, OAT3↑	[30, 60-65]
Pugongying (Taraxaci Herba)	XOD↓, UA↓	[45, 66-68]
Yuganzi (Phyllanthi Fructus)	XOD↓, UA↓, ADA↓, OAT1↑, UARTI↓, GLUT9↓, ALT↓, AST↓, ALP↓, PGE↓, PGE2↓, TNF-α↓, IL-1β↓, IL-6↓, IL-10↓, Nrf2↑, SOD↑	[69–76]
Shanzhuyu (Corni Fructus)	XDH↓, URAT1↓, GLUT9↓, OAT1↑, OAT3↑	[61]
Jinyinhua (Lonicerae Japonicae Flos)	XOD↓, UA↓	[77, 78]
Sangye (Mori Folium)	XOD↓, UA↓	[12, 79, 80]
Yiyiren (Coicis Semen)	XOD↓, UA↓, Cr↓	[81, 82]
Zisuye (Perillae Folium)	XOD↓, UA↓	[13, 34, 83-85]
Heye (Nelumbinis Folium)	XOD↓, UA↓, Cr↓, BUN↓, NLRP3↓, IL-1β↓	[12, 13, 30, 31, 60, 86-94]
Pangdahai (Sterculiae Lychnophorae Semen)	XOD↓, IL-1β↓, IL-6↓, TNF-α↓	[94]
Tianma (Gastrodiae Rhizoma)	XOD↓, UA↓, URAT1↓, OAT1↑	[95-97]
Mugua (Chaenomelis Fructus)	XOD↓, UA↓, Cr↓, BUN↓, UARTI↓, GLUT9↓, OAT1↑, OAT3↑	[98, 99]
Juju (Cichorii Herba)	XOD↓, UA↓, ADA↓, OAT1↑, OAT3↑, UARTI↓, GLUT9↓, PNP↓, CNT2↓, 5'-NT↓, TNF-α↓, IL-1β↓, GD↓, DAO↓, DPEB1↓, sIgA↓, LPS↓	[25, 30, 100-132]
Zhizi (Gardeniae Fructu)	XOD↓, UA↓, OAT1↑, UARTI↓, GLUT9↓, THP↓, ABCG2↓, NLRP3↓, IL-1β↓, PGE2↓, TNF-α↓	[38, 133-141]
Rougui (Cinnamomi Cortex)	XOD↓, NLRP3↓, NLRC4↓, AIM2↓, IL-1β↓, IL-18↓, SOD↑, GSH-Px↑, MDA↓, OAT1↑, ABCG2↑, GLUT9↓, URAT1↓	[142, 143]
Dangshen (Codonopsis Radix)	XOD↓, UA↓, BUN↓, GLUT9↓	[144, 145]
Sharen (Amomi Fructus)	XOD↓, UA↓, OAT1↑, ABCG2↑, URAT1↓, GLUT9↓, IL-1β↓, IL-6↓, TNF-α↓	[146, 147]
Jianghuang (Curcumae Longae Rhizoma)	XOD↓, UA↓, ALT↓, AST↓, Cr↓, BUN↓, MDA↓, IL-1β↓, IL-18↓, NLRP3↓, IκBα↑, NF-κB↓, IL-1β↓, IL-6↓, TNF-α↓, COX-2↓, PGE2↓, CPI↓, TGF-β1↓	[148-155]
Gancao (Glycyrrhizae Radix et Rhizome)	UA↓, Cr↓, BUN↓	[156-158]
Biba (Piperis longi fructus)	PI3K-AKT, MAPK	[159]

"↓" denotes the inhibitory effect of the drug, and "↑" denotes the promotional effect of the drug. OAT2, organic anion transporter 2. OAT3, organic anion transporter 3. Cr, creatinine. BUN, blood urea nitrogen. ALT, alanine aminotransferase. AST, aspartate aminotransferase. IL, interleukin. TNF-α, tumor necrosis factor-α. TLR4, toll-like receptor 4. MDA, malondialdehyde. COX-2, cyclooxygenase-2. ADA, adenosine deaminase. ALP, alkaline phosphatase. PGE, prostaglandin E. Nrf2, nuclear factor-related factor 2. SOD, superoxide dismutase. XDH, xanthine dehydrogenase. PNP, purine nucleoside phosphorylase. CNT2, concentrative nucleoside transporter 2. 5'-NT, 5'-nucleotidase. GD, glutamate dehydrogenase. DAO, diamine oxidase. DPEB1, β-defensin 1. sIgA, secretory immunoglobulin A. LPS, lipopolysaccharide. THP, tissue culture human acute monocytic leukemia cell line. NLRP3, pyrin domain containing 3. NLRC4, NLR family CARD domain containing 4. AIM2, absent in melanoma 2. GSH-Px, glutathione peroxidase. IκBα, nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor alpha. NF-κB, nuclear factor kappa B. CPI, cystatin C. TGF-β1, transforming growth factor-β1. PI3K/AKT, phosphatidylinositol-3-kinase/protein kinase B. MAPK, mitogen-activated protein kinase.

Fructus) [69, 70] were shown to possess XOD inhibitory activity *in vitro*, and their extracts were found to be effective in decreasing the serum UA levels within HUA rats. Additionally, Yuganzi (Phyllanthi Fructus) extracts inhibited ADA activity [70-72], while Shanzhuyu (Corni Fructus) extracts suppressed the mRNA expression of XDH [61].

Jinyinhua (Lonicerae Japonicae Flos) is a Chinese medicine with a long history, and its water-soluble polysaccharide (LJP-1) was able to effectively inhibit XOD activity and significantly reduce serum UA levels [77].

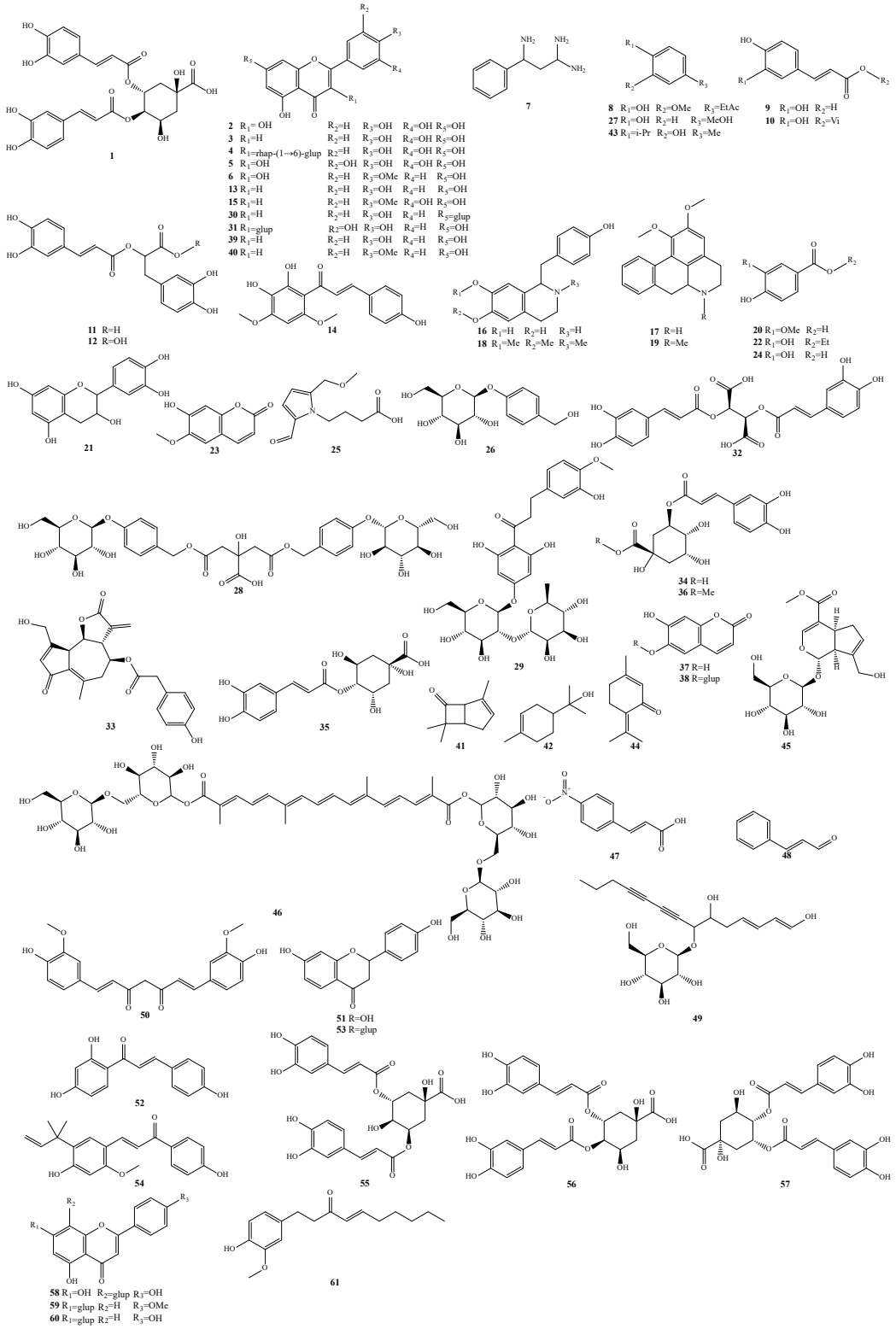

Figure 3 The structures of compounds **1** – **61**

1, 3,4-di-*O*-caffeoylquinic acid. **2**, quercetin. **3**, luteolin. **4**, rutin. **5**, myricetin. **6**, 4'-methoxy-3,5,7-trihydroxyflavone. **7**, 3-phenylpropane-1,1,3-triamine. **8**, dihydroferulic acid. **9**, caffeic acid. **10**, vinyl caffeate. **11**, rosmarinic acid. **12**, methyl rosmarinate. **13**, apigenin. **14**, 2',4'-dimethoxy-4,5',6'-trihydroxychalcone. **15**, diosmetin. **16**, higenamine. **17**, N-nornuciferine. **18**, armepavine. **19**, nuciferine. **20**, vanillic acid. **21**, scopoletin. **22**, ethyl 3,4-dihydroxybenzoate. **23**, catechin. **24**, 3,4-dihydroxybenzoic acid. **25**, 4-(2-formyl-5-(methoxymethyl)-1H-pyrrol-1-yl)butanoic acid. **26**, gastrodin. **27**, p-hydroxybenzyl alcohol. **28**, parishin A. **29**, neohesperidin. **30**, apigenin 7-*O*-glucoside. **31**, quercitrin. **32**, cichoric acid. **33**, lactucopicrin. **34**, chlorogenic acid. **35**, cryptochlorogenic acid. **36**, methyl chlorogenic acid. **37**, esculetine. **38**, esculin hydrate. **39**, apigenin. **40**, acacetin. **41**, filifolone. **42**, α-terpineol. **43**, thymol. **44**, piperitenone. **45**, geniposide. **46**, crocin. **47**, 4-nitrocinnamic acid. **48**, cinnamaldehyde. **49**, lobetyolin. **50**, curcumin. **51**, liquiritigenin. **52**, isoliquiritigenin. **53**, liquiritin. **54**, licochalcone A. **55**, isochlorogenic acid A. **56**, isochlorogenic acid B. **57**, isochlorogenic acid C. **58**, puerarin. **59**, acacetin 7-*O*-glucoside. **60**, apigenin 7-*O*-glucoside. **61**, 6-shogaol.

Additionally, compounds such as methyl 3, 4-di-*O*-caffeoylquinic acid (**1**), quercetin (**2**), luteolin (**3**), and other bis-caffeoylquinic acid and flavonoids have been shown to exhibit XOD inhibitory activity [78].

The extracts of Sangye (Mori Folium) demonstrated inhibitory effects on XOD *in vitro* [12]. Flavonoid compounds, such as rutin (**4**), myricetin (**5**), quercetin (**2**), and 4'-methoxy-3,5,7-trihydroxyflavone (**6**) were capable of decreasing effectively the serum UA levels in HUA rats. The proposed mechanism might be involved in the inhibition of hepatic XOD, affecting the pentose phosphate pathway and consequently led to a decrease in UA synthesis [79, 80].

The compounds, such as 3-phenylpropane-1,1,3-triamine (**7**) and dihydroferulic acid (**8**) isolated from Yiyiren (Coicis Semen) could both significantly reduce the inhibition of xanthine oxidation, exhibit potent XOD inhibitory activity [81, 82], and reduce the serum UA and Cr levels in HUA rats [82].

The compounds, such as caffeic acid (**9**), vinyl caffeate (**10**), rosmarinic acid (**11**), methyl rosmarinate (**12**), apigenin (**13**), and 2',4'-dimethoxy-4,5',6'-trihydroxychalcone (**14**) isolated from Zisuye (Perillae Folium), have been identified as possessing significant XOD inhibitory activity [13, 34, 83, 84]. These compounds have also demonstrated the capability to reduce serum UA levels in HUA rats [34, 83, 85].

Heye (Nelumbinis Folium) is rich in alkaloids and flavonoids, and its extracts include diosmetin (**15**), higenamine (**16**), N-nornuciferine (**17**), armepavine (**18**), and nuciferine (**19**). These compounds demonstrated significant XOD inhibitory activity and antioxidant activity [12, 13, 30, 31, 60, 86-89].

The ethyl acetate extracts of Pangdahai (Sterculiae Lychnophorae Semen) exhibited significant XOD inhibitory activity. The compounds identified, including vanillic acid (**20**), scopoletin (**21**), ethyl 3,4-dihydroxybenzoate (**22**), catechin (**23**), 3,4-dihydroxybenzoic acid (**24**), caffeic acid (**9**), and 4-(2-formyl-5-(methoxymethyl)-1H-pyrrol-1-yl) butanoic acid (**25**), demonstrated considerable inhibitory effects [94].

Gastrodin (**26**) [95], p-hydroxybenzyl alcohol (**27**), and parishin A (**28**) which are constituents of Tianma (Gastrodiae Rhizoma), have demonstrated inhibitory effects on XOD [96]. Flavonoids presented in Mugua (Chaenomelis Fructus) have been shown to effectively reduce XOD activity, and the main active components of these compounds are oligomeric proanthocyanidins, neohesperidin (**29**), apigenin 7-*O*-glucoside (**30**), and quercitrin (**31**) [98].

The extracts of Juju (Cichorii Herba) showed a significant uric acid-lowering effect [30, 101-118]. Key compounds with notable XOD enzyme inhibitory activity include cichoric acid (**32**) [114, 119], lactucopicrin (**33**) [106, 116], chlorogenic acid (**34**) [118], cryptochlorogenic acid (**35**) [118], and methyl chlorogenic acid (**36**) [118]. Furthermore, esculetine (**37**) [120], esculin hydrate (**38**), luteolin (**3**), and quercetin (**2**) are the active compounds in Juju (Cichorii Herba) with potential XOD inhibitory and uric acid-lowering properties [106, 114].

While Juhua (Chrysanthemi Flos) extracts showed a significant XDH inhibitory activity [61], luteolin (**3**), apigenin (**39**), and acacetin (**40**) also demonstrate potent XOD enzyme inhibitory effects [65]. Additionally, four monoterpenes of filifolone (**41**), α-terpineol (**42**), thymol (**43**), and piperitenone (**44**) also strongly associated with XOD inhibition [65].

Furthermore, geniposide (**45**) and crocin (**46**) [38, 133-139] extracted from Zhizi (Gardeniae Fructus), 4-nitrocinnamic acid (**47**) and cinnamaldehyde (**48**) extracted from Rougui (Cinnamomi Cortex) [142], lobetyolin (**49**) [144] from Dangshen (Codonopsis Radix), vanillic acid (**20**) [146, 147] from Sharen (Amomi Fructus), and curcumin (**50**) [148, 149] from Jianghuang (Curcumae Longae Rhizoma), all demonstrated significant XOD inhibitory activity.

3.2 Chinese medicines for promoting the excretion of UA

Four flavonoids identified in Gancao (Glycyrrhizae Radix et Rhizoma): liquiritigenin (**51**), isoliquiritigenin (**52**), liquiritin (**53**), and licochalcone A (**54**), have been shown to significantly reduce the serum UA levels in HUA rats [156]. Specifically, liquiritigenin was able to inhibit the reabsorption of UA in renal tubules by interacting with UA transporters, thereby enhancing UA excretion, and subsequently lowering its concentration in HUA rats [157]. Isoliquiritigenin could reduce UA, Cr, and BUN levels in HUA rats [158], further decreasing overall UA.

Study has demonstrated that Heye (Nelumbinis Folium) extracts can effectively alleviate HUA by regulating UA synthesis and transport, and decrease inflammation levels [88]. Furthermore, it could regulate the expression of urate transporter mRNA in renal tissues, and reduce the UA production. The main active compound nuciferine (**19**) in Heye (Nelumbinis Folium), has been shown to regulate disordered tyrosine metabolism, citrate metabolism, arginine and proline metabolism, glycolysis metabolism, and gluconeogenesis [90], leading to the serum levels of UA, Cr, and BUN reduction. This effect is achieved by altering the gut microbiota and kidney protein levels, while simultaneously enhancing the fractional excretion of UA [91].

Compounds extracted from Juju (Cichorii Herba), such as esculin hydrate (**38**), chlorogenic acid (**34**), cichoric acid (**32**), isochlorogenic acid A(**55**)/B(**56**)/C(**57**) and 13,14-seco-stigma-5(6),14(15)-diene-3α-ol [121], have demonstrated a significant uric acid-lowering effect. Studies have indicated that Juju (Cichorii Herba) extracts inhibited the pentose phosphate and glycolytic pathways [111], while simultaneously upregulating the expression of

OAT1 [122] and OAT3 [123]. Additionally, it downregulated the expression of ABCG2 [25, 124, 125], GLUT9 [25, 100, 122, 126], and URAT1 [25, 100, 119], thereby reducing UA production. Furthermore, research has also shown that Juju (Cichorii Herba) extracts can downregulate the mRNA and protein expression of CNT2 in rat jejunum, reduce the activities of 5'-NT [109, 113], ADA [109, 110, 112-114, 116, 117, 123], PNP, and GD [113], thereby decreasing the purine absorption and UA production. Study has demonstrated that the extracts from Juju (Cichorii Herba) can effectively reduce serum concentrations of DAO, thereby ameliorating damage to the intestinal barrier function, maintaining the balance of the intestinal barrier, mitigating abnormal elevations in intestinal DPEB1, and stimulating the secretion of intestinal sIgA [128]. Furthermore, it has been shown to modulate gut microbiota [110, 111, 124, 129], decrease peripheral blood LPS levels [111, 130], reduce intestinal epithelial cell permeability, and consequently influence key enzymes involved in UA metabolism to enhance its excretion [110].

Extracts of Shanzhuyu (Corni Fructus) [61], Zhizi (Gardeniae Fructus) [135, 140], Sharen (Amomi Fructus) [146], and Mugua (Chaenomelis Fructus) [78, 99] have demonstrated the ability to upregulate the expression levels of OAT1 and OAT3, thus accelerating the secretion of UA. Additionally, these extracts could inhibit the expression of GLUT9 and URAT1, leading to the reduction of reabsorption of UA and its excretion promotion. Extracts from Zhizi (Gardeniae Fructus) have also been found to downregulate the expression of ABCG2 [140]. Furthermore, extracts from Yuganzi (Phyllanthi Fructus) [71] and Fuling (Poria) [40-42] could upregulate the expression of OAT1 and downregulate the expression of GLUT9 and URAT1. Moreover, Fuling (Poria) extracts may enhance the expression of OAT1, OAT3, ABCG2, and organic cation transporter protein 2 (OCT2) [43], while also improving renal function and modulating the gut microbiota composition [39].

Extracts from Juhua (Chrysanthemi Flos) [62, 63], Tiepishihu (Dendrobii Officinalis Caulis) [35], Xihonghua (Croci Stigma) [36], and the essential oil of Rougui (Cinnamomi Cortex) [142] have demonstrated the ability to upregulate renal ABCG2 expression while downregulating the expression of GLUT9 and URAT1, thus enhancing UA excretion and lowering serum UA level. Additionally, Yejuhua (Chrysanthemi Indici Flos) extracts were found to increase the expression levels of OAT1 and OAT3 [61]. The antihyperuricemic effects of Xihonghua (Croci Stigma) extracts may be attributed to its regulation of serum lipids [36], modulation of arginine biosynthesis, glycine, serine, threonine metabolism, and histidine metabolism and the regulation of intestinal flora is closely associated with host metabolism [36, 37]. Moreover, the essential oil of Rougui (Cinnamomi Cortex) [142] can reduce UA level by upregulating OAT1 expression.

Roudoukou (Myristicae Semen) significantly reduced serum UA levels in HUA rats [56]. Similarly, Huangqi (Astragali Radix) [44-46] and Jianghuang (Curcumae Longae Rhizoma) extracts, curcumin (**50**) [149-153], exhibited favorable uric acid-lowering effects, effectively downregulating serum Cr, BUN, ALT, and AST levels. Huangqi (Astragali Radix) extracts have been shown to enhance UA excretion *in vivo* by regulating the expression of transporter proteins in the kidney and ileum [25], modulate metabolic pathways such as linoleic acid and glycerophospholipids, and the intestinal microbiota, thereby increasing the relative abundance of beneficial bacteria and mediating intestinal metabolic pathways associated with UA excretion, thus achieving uric acid-lowering effects [44, 47].

Furthermore, gastrodin (**26**), the principal active constituent of Tianma (Gastrodiae Rhizoma), has been shown to effectively reduce serum UA levels, downregulate renal URAT1 expression, and upregulate OAT1 expression in HUA rats [95-97]. Extracts from Duzhongye (Eucommiae Folium) have demonstrated the ability to modulate UA, Cr, and BUN levels and activities in HUA rats [48], and they can treat HUA by regulating GLUT9 expression [50]. Gouqizi (Lycii Fructus) extracts have been found to significantly decrease serum GLUT9 levels in HUA rats [49] and enhance the expression levels of OAT1 and OAT3 [51]. Gaoliangjiang (Alpiniae Officinarum Rhizoma) extracts can regulate serum Cr and BUN levels, downregulate URAT1 and GLUT9 expression, and reduce hepatic MDA levels in HUA rats [13], thus achieving therapeutic effects. Dangshen (Codonopsis Radix) extracts have been shown to improve UA levels, reduce serum BUN levels, and downregulate hepatic GLUT9 expression in HUA rats [145]. Puerarin (**58**), an extract of Gegen (Puerariae Lobatae Radix), can promote the expression of ABCG2 protein and mRNA [59] and inhibit ALP activity.

3.3 Chinese medicines with anti-inflammatory properties and renal protective effects

In addition to inhibiting UA production and promoting its excretion, certain medications also exhibited anti-inflammatory properties, which contributing to alleviate the pathological conditions in the kidneys, thus providing renal protection and therapeutic support.

For example, studies have demonstrated that the Heye (Nelumbinis Folium) can reduce serum and renal IL-1β levels, and alleviate renal inflammatory pathology by inhibiting the toll-like receptor 4/myeloid differentiation factor 88/nuclear factor kappa B (TLR4/MyD88/NF-κB) signaling pathway and NLRP3 inflammasomes [92, 93]. Furthermore, in a HUA inflammatory cell model, an extract from Pangdahai (Sterculiae Lychnophorae Semen) inhibited the PI3K/AKT/NF-κB signaling pathway, resulting in a significant decrease in the secretion of IL-1β, IL-6, and TNF-α in inflammatory cell model. This mechanism

effectively mitigated uric acid-induced inflammation and protected human kidney-2 cells (HK-2) in a high UA environment [94].

Luteolin (**3**), acacetin 7-*O*-glucoside (**59**), and apigenin 7-*O*-glucoside (**60**) in Juhua (Chrysanthemi Flos) have demonstrated a reduction in IL-1β levels in THP-1 cells, suggesting their potential role as key components responsible for the anti-inflammatory effects of Juhua (Chrysanthemi Flos) [64].

Yuganzi (Phyllanthi Fructus) extracts have been shown to effectively inhibit the elevation of serum ALT, AST, and ALP, thereby improving liver histopathology in animal models of liver injury [73]. It inhibited the production of the inflammatory mediator PGE [74, 75] in HUA rats joint tissue, decreased the levels of serum TNF-α, IL-1β, IL-6, IL-10, and PGE2, and upregulated the expression of Nrf2 in cytoplasm of synovial cells, and enhancing the activity of SOD [71, 75, 76]. Yuganzi (Phyllanthi Fructus) extracts also inhibited blood sedimentation rate in HUA rats [75, 76].

Duzhongye (Eucommiae Folium) extracts have been shown to ameliorate HUA and mitigate kidney injury and inflammation by regulating serum IL-6, TNF-α, and TLR4 [50]. Gouqizi (Lycii Fructus) extracts significantly decreased the serum levels of TNF-α and IL-1β in HUA rats [49]. Biba (Piperis longi fructus) primarily exerts its uric acid-lowering effect through modulation of the PI3K-AKT signaling pathways and MAPK signaling pathways [159].

Zhizi (Gardeniae Fructus) extracts inhibited the activation of the TLR4/TANK-binding kinase 1 (TBK1)/IκB kinase-ε (IKKε) signaling pathway [141], suppressed the expression of the TLR4/TLR2/MyD88/NF-κB and neutrophilic alkaline phosphatase 3/apoptosis-associated speck-like protein containing a CARD/cysteinyl aspartate specific proteinase 1 (NALP3/ASC/caspase-1) [138] signaling pathways, and inhibited the activation of NLRP3 inflammasomes. This inhibition led to a reduction in the production of renal inflammatory factors, such as IL-1β, PGE2, and TNF-α, and a decrease in the expression levels of these inflammatory factors in renal tissues [137, 138].

Juju (Cichorii Herba) extracts and chicoric acid effectively inhibited the TLR4/NF-κB/NLRP3 signaling pathway, thereby reducing the release of serum IL-1β, alleviating the inflammatory response mediated by the LPS/TLR4 axis, and promoting UA excretion [130, 131]. Additionally, Ganjiang (Zingiberis Rhizoma) extract, particularly 6-shogaol (**61**), significantly lowered serum UA levels and the concentrations of inflammatory markers, such as IL-1β, IL-6, TNF-α, and COX-2, in HUA rats [132].

Rougui (Cinnamomi Cortex) extracts effectively inhibits the activation of inflammasome in cells, such as NLRP3, NLRC4, and AIM2, as well as the secretion of IL-1β and IL-18, positioning it as a promising therapeutic agent for inflammatory diseases [143]. Moreover, essential oil of Rougui (Cinnamomi Cortex) enhanced the levels of SOD and GSH-Px, while simultaneously reducing MDA levels, thereby augmenting the organism's antioxidant capacity, and alleviating renal inflammation. Roudoukou (Myristicae Semen) extracts could decrease the level of TNF-α [56]. Puerarin (**58**), derived from Gegen (Puerariae Lobatae Radix), suppresses ALP activity, contributing to its nephroprotective effects [59]. Sharen (Amomi Fructus) demonstrated therapeutic benefits by downregulating the expression levels of IL-1β, IL-6, and TNF-α in hepatic or renal tissue [146].

Curcumin (**50**), an extract of Jianghuang (Curcumae Longae Rhizoma), restored SOD and GSH-Px activities, lowered MDA and CPI levels, and decreased serum levels of IL-1β and IL-18. It also inhibited NLRP3 inflammasome signaling [154]. In cell experiments, curcumin prevented IκBα degradation, NF-κB activation, and the production of IL-1β, IL-6, TNF-α, COX-2, and PGE2 in THP-1-derived macrophages stimulated by monosodium urate (MSU), and suppressed NLRP3 inflammatory activity [155]. Curcumin also reduced the expression level of TGF-β1 [151-153].

4 Discussion

As reported in the "2021 White Paper on the prevalence and trends of HUA and Gout in China" [160], over 170 million people suffer from HUA, among them, 14 million diagnosed with gout. Notably, patients with HUA and gout are prone to younger age groups. Now, HUA is ranked the fourth most common metabolic disorder after hypertension, hyperlipidemia, and hyperglycemia. Current treatments are mainly focusing on the synthetic drugs, such as inhibiting UA production with Allopurinol and Febuxostat, and increasing UA excretion with Benzbromarone. Similar to the "Three Highs" (hypertension, hyperlipidemia, and hyperglycemia), HUA also requires long-term medication, which poses higher demands for drug safety, while the drugs commonly used in clinical for HUA often cause severe side effects. Thus, the exploration of Chinese medicine with medicine food homology with UA lowering activity is of great significance in effectively treating HUA. This paper reviews literature of Chinese medicine with medicine food homology with UA-lowering activity over the past 22 years. We found that 43 extracts from these substances can significantly reduce UA levels by inhibiting its synthesis, increasing its excretion, or reducing renal inflammation.

Research on the UA-lowering effects of Chinese medicine with medicine food homology mainly focuses on their extracts, while there is relatively little research on their specific active components. A total of 61 active compounds were reported from 2002 to 2024, primarily polyphenols (29 species) and flavonoids (17 species). Studies consistently showed that these active compounds

have significant UA-lowering activity [58, 161-165]. However, some compounds and drugs have only been tested for XOD inhibitory activity *in vitro*, and there is lack of research on their UA-lowering efficacy *in vivo*. Future research should focus on enhancing the *in vivo* UA-lowering activity of these compounds to lay a strong foundation for developing novel UA-lowering agents.

Most studies on the treatment of HUA with medicine-food homology have focused on the regulation of XOD activity, the expression of transporters related to UA excretion and reabsorption, anti-inflammatory, the renal protective effects, and the gut microbiota modulation. While some research has explored its pharmacological activities and efficacy, the specific mechanisms underlying its treatment of HUA remain unclear, and further investigation is still required. Currently, the application of medicine food homology in treating HUA is mainly limited to TCM prescriptions and health products. Standardized protocols for extracting or synthesizing active substances are still lacking, which hinders its widespread use as a UA-lowering agent. However, these compounds with strong XOD inhibitory activity show significant potential in treating HUA, which makes it promising in gradually replacing allopurinol with severe side effects. Due to their dual-purpose nature, these compounds can also serve as dietary additives, aiding in the prevention and management of HUA.

5 Conclusion

HUA has become a significant disease that threatened human health nowadays. Currently, anti-hyperuricemia drugs often cause severe side effects, while Chinese herbal medicines with medicine-food homology have received increasing attention. This review focuses on the UA-lowering efficacy of these Chinese herbal medicines. Among 108 well-known Chinese medicines with medicine-food homology, 43 kinds of Chinese medicines with notable effects were identified, 61 kinds of active compounds, mainly polyphenols and flavonoids, with UA-lowering activities were listed. The mechanisms behind these effects are explained, providing a strong basis for developing safer and more effective therapies. Moreover, the mechanism underlying the UA-lowering effect of these Chinese herbal medicines with medicine-food homology and their active constituents was comprehensively summarized. This work is expected to lay a foundation for the exploration and development of novel and efficacious UA-lowering medications.

Fundings

National Key R & D Program of China (2021YFD 1600301 and 2021YFD1600105), and Hunan Provincial Key Research and Development Project (2023NK2041).

Competing interests

The authors declare no conflict of interest.

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药食同源中药降尿酸活性及其作用机制的系统研究

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【摘要】目的 通过总结具有药食同源性中药的降尿酸作用与机制，为高尿酸血症（HUA）治疗药物的研发提供新的思路。**方法** 本文通过中国知网 (CNKI)、PubMed、ScienceDirect、谷歌学术等平台筛选收集 2002 年 12 月 15 日至 2024 年 8 月 10 日期间关于药食同源中药防治 HUA 研究的相关文献。随后，评估这些中药及其提取物以及活性化合物对 HUA 的影响。**结果** 共收集相关文献 148 篇，包括具有抗 HUA 活性的 43 种中药和 61 种活性化合物。其中 41 种中药均能够抑制黄嘌呤氧化酶活性，从而抑制尿酸生成；22 种中药有助于尿酸排泄；15 种中药可降低机体炎症水平，起到肾脏保护的作用。值得注意的是，多酚类和黄酮类化合物是降尿酸的关键活性成分。**结论** 本研究系统分析总结了药食同源中药的降尿酸作用及其机制，为其作为治疗 HUA 药物的研发奠定基础。

【关键词】高尿酸血症；药食同源；中药；活性成分；提取物